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ELECTROPHYSIOLOGIC STUDIES

Ventricular Response to Atrial Fibrillation: Role of Atrioventricular Conduction Pathways

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Irregularity of the ventricular rhythm is a hallmark of patients with atrial fibrillation, yet the genesis of the irregularity is not yet fully understood. The role of the atrioventricular (AV) node in determining the irregularity of the ventricular response to atrial fibrillation was investigated by comparing the frequency distributions of the atrial (AA) and the ventricular (RR) intervals.

Atrial electrograms and surface electrocardiographic leads were recorded during sustained atrial fibrillation in 12 patients with conduction over the AV node. The scaling factor (mean RR interval/mean AA interval) quantified the ability of the conduction pathway to scale the atrial input to a slower ventricular response and ranged from 2.55 to 5.92 (mean ± SD 3.77 ± 0.92). The coefficient of variation (SD/mean) measured the relative variability of the AA and RR interval distributions. The atrial and ventricular coefficients of variation were not significantly different (0.20 ± 0.04 versus 0.21 ± 0.03, $p > 0.27$).

Similar recordings were analyzed in six patients with conduction over an accessory AV pathway. The scaling factor ranged from 1.54 to 2.46 (2.02 ± 0.39) and, as was the case for patients with conduction over the AV node, the atrial and ventricular coefficients of variation did not significantly differ (0.24 ± 0.08 versus 0.27 ± 0.10, $p > 0.6$). For both groups of patients, ventricular variability and the maximal RR intervals were predicted by the product of the scaling factor and either atrial variability or maximal AA intervals, respectively.

This study adds direct observation of atrial events to a body of data that suggests that the irregularity of the ventricular response to atrial fibrillation is primarily a consequence of the irregularity inherent in the atrial activity. This relation was observed regardless of the nature of the AV conduction pathway, suggesting that no special properties of the AV node need to be considered to account for the irregularity of the ventricular response to atrial fibrillation.

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Irregularity of the ventricular rhythm as a consequence of atrial fibrillation has long been recognized (1), yet its explanation remains unclear (2). Numerous studies focusing on concealed conduction within the atrioventricular (AV) node (3–6) and statistical measures of the RR intervals (3,7–14) have been undertaken, with conflicting conclusions as to the factors determining the ventricular response (2). More recently, studies relating the electrophysiologic properties of AV conducting pathways to the ventricular response during atrial fibrillation have been performed (15–20), but there remains controversy as to the exact cause of the ventricular irregularity (21,22).

There is evidence that the role of the AV node during atrial fibrillation is that of transmitting the random, irregular pattern of atrial excitation to the ventricles at a scaled-down rate (12,23,24); however, no published study has quantitatively compared the variability of the ventricular response with any variability present in the atrial excitation intervals.

In this study, both atrial and ventricular rhythms in a group of patients with atrial fibrillation were examined. Statistical techniques were used in investigating the relation between the patterns of atrial excitation and the ventricular response to atrial fibrillation. Specifically, the coefficient of variation allowed comparison of the irregularity of the atrial and ventricular rhythms as fractions of their respective mean cycle lengths. These same techniques were then applied in a group of patients with conduction over an accessory AV pathway during atrial fibrillation, and the results were com-

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Table 1. Clinical Characteristics in 18 Patients

Patient	Age(yr)/ Gender	Dx	Pathway	Atrial Fibrillation	Medications
1	68M	CAD	AVN	Chronic	None
2	72M	CAD	AVN	Chronic	None
3	60M	CAD	AVN	Chronic	Digoxin
4	71F	CHD	AVN	Chronic	Digoxin, verapamil
5	58M	CAD	AVN	Chronic	Digoxin
6	79M	CAD	AVN	Chronic	Diltiazem, metoprolol
7	50M	CM	AVN	Chronic	Digoxin, verapamil
8	64M	AS	AVN	Chronic	Digoxin, diltiazem
9	25M	WPW	AVN	Induced	None
10	26M	WPW	AVN	Induced	None
11	30M	WPW	AVN	Induced	None
12	47M	WPW	AVN	Induced	None
13	19M	WPW	AP	Induced	None
14	53F	WPW	AP	Induced	None
15	25M	WPW	AP	Induced	None
16	65F	WPW	AP	Induced	None
17	48M	WPW	AP	Induced	None
18	28M	WPW	AP	Induced	None

AP = accessory AV pathway; AS = aortic stenosis; AVN = AV node; CAD = coronary artery disease; CHD = congenital heart disease; CM = cardiomyopathy; Dx = diagnosis; F = female; M = male; WPW = Wolff-Parkinson-White syndrome.

pared with those from the patients with conduction over the AV node.

Methods

Criteria for admission to the study. Patients undergoing either hemodynamic cardiac catheterization or electrophysiologic study who demonstrated either chronic or induced sustained atrial fibrillation were considered eligible for inclusion in this study. The study was limited to those patients who exhibited either type I or type II atrial fibrillation and therefore had discrete atrial electrograms (25) with AV conduction exclusively over the AV node *or* exclusively over an accessory AV pathway. Patients with type III fibrillation (25) were excluded because of difficulty in accurately determining atrial intervals in the absence of discrete electrograms. The study was approved by the institution's committee on human research, and written informed consent was obtained from all patients before study.

Study patients (Table 1). A total of 18 patients were studied, and their clinical diagnoses are summarized in Table 1. Twelve patients (11 men, 1 woman), ranging in age from 25 to 79 years (mean 55), had conduction over the AV node during atrial fibrillation. Six patients (four men, two women), ranging in age from 19 to 65 years (mean 40), with Wolff-Parkinson-White syndrome had conduction over an accessory AV pathway during atrial fibrillation. All 10 patients with induced atrial fibrillation, including 4 with conduction over the AV node and 6 with conduction over an accessory pathway, were free of all cardioactive medications for at least five drug half-lives before study. Six of the eight patients with chronic atrial fibrillation (all with conduction over the AV node) were taking various negative dromotropic medications.

Data acquisition. Continuous recordings of 2 to 5 (mean 4) min duration were made during atrial fibrillation. A bipolar high right atrial electrogram with a 1 cm interelectrode spacing (USCI), as well as surface electrocardiographic (ECG) leads I, II and V_1, were amplified and filtered using a physiologic recorder (Honeywell VR-16; Electronics for Medicine, Honeywell Inc.) as previously described for our laboratory (26,27). The intraatrial signals were filtered using a passband of 30 to 500 Hz, whereas the surface leads were filtered using a passband of 0.05 to 5,000 Hz. Signals were recorded on frequency-modulated (FM) tape (Honeywell 101). In three cases the patients were studied at another laboratory, and the signals were amplified and filtered using a similar Honeywell VR-16 recorder, recorded on FM tape using a Honeywell 5600E and then rerecorded using a Vetter model B FM tape recorder (A. R. Vetter) for transport.

Preprocessing. One surface lead and the high right atrial electrogram were played back simultaneously from tape through an antialiasing filter with a cutoff frequency of 120 Hz. The signals were amplified and then digitized at 1,200 Hz using a Masscomp MCS-563 computer system (Massachusetts Computer). These signals were then reduced to an effective sampling rate of 240 Hz by extracting every fifth data point while still satisfying the Nyquist sampling criterion (28).

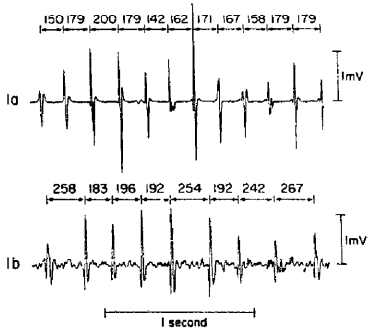

Figure 1. High right atrial electrograms during atrial fibrillation with detected AA intervals marked. As defined by Wells et al. (25), type I fibrillation (Ia) is characterized by discrete complexes separated by an isoelectric baseline whereas type II fibrillation (Ib) is characterized by discrete complexes with perturbations of the baseline between complexes. Type III fibrillation fails to demonstrate either discrete complexes or isoelectric intervals, and thus the measurement of AA intervals is less certain. All AA interval measurements are in milliseconds.

Atrial and ventricular event detection. The digitized high right atrial signals were scanned for discrete electrograms with use of an adaptive threshold detection algorithm previously described by our laboratory (27). Patients in the study were restricted to those exhibiting either type I or type II fibrillation as described by Wells et al. (25), and thus by definition had discrete atrial electrograms. Atrial cycle lengths were measured as the intervals between successive discrete complexes (Fig. 1). A graphic display of the atrial signal, with detected electrographic complexes marked, was generated on-line and each detected complex was manually confirmed.

R waves in the digitized surface ECG lead were detected using a differentiation-squaring algorithm adapted from that of Hamilton and Tompkins (29). A graphic display of the surface lead signal with detected R waves marked generated on-line, and each detected R wave was manually confirmed.

Interval histograms. Frequency distribution histograms were generated for both atrial and ventricular cycle lengths, with a class width of 20 and 50 ms, respectively (Fig. 2). The maximal AA and RR intervals were defined as the 95th percentile points of the atrial and ventricular cycle length distributions, respectively.

The frequency of occurrence of RR intervals longer than the mean has previously been observed to fall off along an exponential decay curve reminiscent of the Poisson distribution (10). This result would cause a plot of the natural logarithm of interval frequency versus interval length to appear as a straight line with a negative slope. To quantify the shape of the right-hand tail of both the AA and RR distributions, the normalized histogram was plotted on a semilogarithmic scale. Regression lines were calculated relating the natural logarithm of interval frequency to the interval length, and correlation coefficients were measured.

Scaling factors and coefficients of variation. The scaling factor, which was defined as the ratio of mean ventricular to mean atrial cycle length, quantified the ability of the AV conduction pathway to transform its rapid atrial input into a slower ventricular response. To measure the relative variability of a distribution (i.e., normalized to its mean), the coefficient of variation was calculated as the ratio of the standard deviation of the AA or RR interval distribution to its mean (13,14,30). It was hypothesized that, if the role of the AV conduction system during atrial fibrillation were solely that of scaling the atrial input to a slower ventricular response, then the absolute variability inherent in the atrial rhythm (SD_{AA}, the standard deviation of the AA interval distribution) would be similarly scaled to produce the absolute variability in the ventricular rhythm (SD_{RR}, the standard deviation of the RR interval distribution), and the coefficients of variation would not differ between the atria and ventricles. Similarly, the maximal atrial intervals would be scaled to produce the maximal ventricular intervals.

The term "scaling" has been used by several authors [12,23,24] to describe the role of the AV node in determining the ventricular response to atrial fibrillation. In this study,

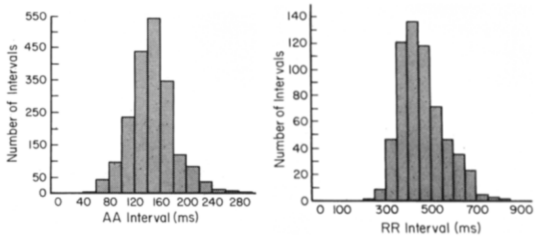

Figure 2. Case 9. Atrial and ventricular interval histograms. The horizontal axes are not equivalent but were instead set such that the full scale RR interval is equal to the scaling factor multiplied by the full scale AA interval. The vertical scales were set automatically by the computer to provide maximal vertical resolution. This choice of scales demonstrates the similarity in shape of the two distributions.

Table 2. Results in 18 Patients

Patient	Pathway	Mean AA (ms)	Mean RR (ms)	SD_{AA} (ms)	SD_{RR} (ms)	SF	CV_{AA}	CV_{RR}
1	AVN	151.3	895.0	23.1	186.9	5.92	0.153	0.209
2	AVN	197.7	794.2	36.5	148.7	4.02	0.185	0.187
3	AVN	217.7	811.7	37.2	118.5	3.73	0.171	0.146
4	AVN	286.3	857.3	61.4	205.6	2.99	0.214	0.240
5	AVN	274.0	1003.6	39.4	248.5	3.66	0.144	0.248
6	AVN	252.3	823.3	45.8	168.6	3.26	0.182	0.205
7	AVN	163.5	576.8	25.5	148.1	3.53	0.156	0.257
8	AVN	246.4	1151.8	55.5	254.6	4.67	0.225	0.221
9	AVN	146.2	461.2	33.6	97.9	3.15	0.230	0.212
10	AVN	140.5	358.2	39.1	80.2	2.55	0.279	0.224
11	AVN	147.2	670.4	35.4	165.6	4.55	0.240	0.247
12	AVN	160.0	507.4	28.0	89.2	3.17	0.175	0.176
13	AP	170.0	385.1	53.5	71.8	2.26	0.315	0.186
14	AP	174.8	269.8	35.0	80.8	1.54	0.200	0.300
15	AP	155.4	369.2	28.9	109.3	2.37	0.186	0.296
16	AP	105.7	259.9	20.8	37.4	2.46	0.197	0.144
17	AP	172.7	291.3	63.2	69.9	1.69	0.366	0.240
18	AP	183.8	326.0	37.8	138.3	1.77	0.205	0.424

CV_{AA} = SD_{AA}/mean AA = atrial coefficient of variation; CV_{RR} = SD_{RR}/mean RR = ventricular coefficient of variation; SD = standard deviation; SF = mean RR/mean AA = scaling factor. Other abbreviations as in Table 1.

the use of the term "scaling" is meant only to maintain historical consistency rather than imply an assumption as to the precise mechanism of AV node function.

Linear regression and statistical tests of "best fit" lines. Plots were drawn of the ventricular variability (SD_{RR}) versus the scaled atrial variability (scaling factor multiplied by SD_{AA}) and of the maximal (95th percentile) RR interval versus the scaled maximal AA interval (scaling factor multiplied by the 95th percentile AA interval). Regression lines were calculated using the method of least-squares. The slope and intercept of the best fit line were then tested against the slope and intercept of the identity line (1.0 and 0.0, respectively) using the Student's *t* test (31).

Results

Patients With Conduction Over the AV Node (Table 2)

By study design, all atrial electrograms were indicative of either type I (n = 4) or type II (n = 8) fibrillation (25), exhibiting discrete complexes of variable timing and configuration separated by either an isoelectric baseline or one of varying degrees of perturbation (Fig. 1). The mean atrial cycle length ranged from 140 to 286 ms (mean ± SD 199 ± 54 ms). In all patients, the ventricular response to atrial fibrillation was markedly irregular, with mean cycle lengths ranging from 358 to 1,152 ms (743 ± 234 ms).

Interval histograms (Fig. 2). In all patients, both the AA and RR intervals were unimodally distributed. Qualitatively, patients with longer mean ventricular cycle lengths were observed to have wider interval histograms, reflecting an increase in the absolute variability of the RR intervals. The semilogarithmic plots of the right-hand tail of the distributions were all linear, as demonstrated by correlation coefficients ranging from −0.907 to −0.996 (mean −0.967) for the AA interval distributions and correlation coefficients ranging from −0.860 to −0.981 (mean −0.940) for the RR interval distributions. All of the correlation coefficients were statistically significant (all $p < 0.005$). This linearity demonstrated that the right-hand tails of both the AA and RR interval distributions had similar shapes, following an exponential decay curve.

Scaling factors and coefficients of variation. The scaling factors (mean RR/mean AA) ranged from 2.55 to 5.92 (3.77 ± 0.92) and were found to relate both atrial variability and maximal AA intervals to ventricular variability and maximal RR intervals, respectively (see next section). The atrial coefficients of variation (SD_{AA}/mean AA) ranged from 0.14 to 0.28 (0.20 ± 0.04). The ventricular coefficients of variation (SD_{RR}/mean RR) ranged from 0.14 to 0.26 (0.21 ± 0.03). There was no significant difference between the atrial and ventricular coefficients of variation ($p > 0.27$) suggesting that scaling of atrial variability can account for ventricular variability.

Maximal intervals. In the 12 patients, with conduction over the AV node, the observed maximal RR interval ranged from 508 to 1,634 ms (1,026 ± 338 ms). The product of the scaling factor and maximal AA interval ranged from 510 to 1,597 ms (988 ± 313 ms). Similar to the comparison of coefficients of variation described, these were not significantly different ($p > 0.5$), suggesting that scaling of the

maximal atrial intervals is sufficient to account for the maximal ventricular intervals.

Patients With Conduction Over an Accessory Pathway (Table 2)

To further investigate the role of the AV conduction system in determining the irregularity of the ventricular response to atrial fibrillation, the group of six patients with conduction over an accessory pathway was examined and compared with the patients with conduction over the AV node. By study design, the atrial electrograms were all indicative of either type I ($n = 2$) or type II ($n = 4$) fibrillation. The mean atrial cycle length ranged from 106 to 184 ms (160 ± 28 ms). This length was not significantly shorter ($p > 0.15$) than the mean atrial cycle length (199 ± 54 ms) found in patients with conduction over the AV node. The ventricular response was irregular, with mean cycle lengths ranging from 260 to 385 ms (317 ± 52 ms).

Interval histograms. The frequency histograms of the atrial and ventricular cycle lengths were similar to those found for the group with conduction over the AV node except that in one patient the RR distribution was bimodal. The semilogarithmic plots of the right-hand tail of the distributions were all linear. The correlation coefficients of these plots for the AA intervals ranged from −0.916 to −0.991 (mean −0.962), and those for the RR intervals ranged from −0.834 to −0.984 (mean −0.915). All of the correlation coefficients were statistically significant (all $p < 0.05$). This linearity again demonstrated that the right-hand tail of both the AA and RR interval distributions had similar shapes, following an exponential decay curve.

Scaling factors and coefficients of variation. The scaling factors ranged from 1.54 to 2.46 (2.02 ± 0.39). This range was significantly ($p < 0.0005$) lower than the mean scaling factor (3.77 ± 0.92) found in patients with conduction over the AV node, consistent with the fact that the accessory pathway is less effective at scaling down the atrial input than is the AV node.

The atrial coefficients of variation ranged from 0.19 to 0.37 (0.24 ± 0.08), and the ventricular coefficients of variation from 0.14 to 0.42 (0.27 ± 0.10). As for patients with conduction over the AV node, there was no significant difference between the atrial and ventricular coefficients of variation ($p > 0.6$), suggesting that scaling of the atrial variability can account for the ventricular variability regardless of the nature of the AV conduction pathway.

Maximal intervals. The maximal RR interval ranged from 329 to 571 ms (463 ± 93 ms). As would be expected, this interval was significantly ($p < 0.0005$) shorter than the maximal RR interval (1,026 ± 338 ms) observed in patients with conduction over the AV node. The product of scaling factor and maximal AA interval ranged from 327 to 565 ms (444 ± 90 ms). As was the case for patients with conduction over the AV node, there was not a statistically significant difference ($p > 0.7$) between the scaled maximal AA intervals and the maximal RR intervals, again suggesting that scaling of the maximal atrial cycle lengths is sufficient to account for the maximal RR intervals regardless of the nature of the AV conduction pathway.

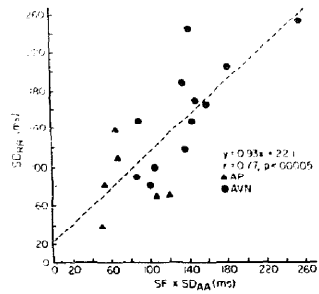

Figure 3. Prediction of ventricular variability (SD_{RR}) by atrial variability (SD_{AA}) related by the scaling factor (SF). The regression line is not significantly different from the line of identity ($p > 0.4$), suggesting that atrial variability during atrial fibrillation can account for ventricular variability. AP = accessory AV pathway; AVN = AV node; SD = standard deviation.

Atrial variability and maximal atrial cycles as predictors of ventricular variability and maximal ventricular cycles. To illustrate the relation between atrial and ventricular variability, a plot of the product of the scaling factor and the standard deviation of the AA interval distribution versus the standard deviation of the RR interval distribution for both groups of patients is shown in Figure 3. The two quantities are strongly correlated ($r = 0.77$, $p < 0.0005$) and the regression line is not significantly different from the line of identity ($p > 0.4$). This predictability reflects the equality of the atrial and ventricular coefficients of variation previously described. In Figure 3 the data from both groups of patients lie along the same line regardless of the nature of the AV conduction pathway.

To illustrate the relation between the maximal atrial and ventricular cycles, a plot of the product of the scaling factor and the maximal AA intervals versus the maximal RR interval for both groups of patients is shown in Figure 4. There is again a strong correlation ($r = 0.98$, $p < 0.0005$) and the regression line is not significantly different from the line of identity ($p > 0.4$). Again, the data from both groups of patients lie along the same line regardless of the nature of the AV conduction pathway.

Discussion

Previous studies. Early work by Arnoldi (32) and then Soderstrom (7) focused on the existence of a number of

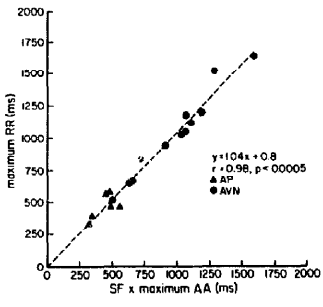

Figure 4. Prediction of the maximal (95th percentile) RR interval by the maximal (95th percentile) AA interval related by the scaling factor (SF). The regression line is not significantly different from the line of identity ($p > 0.4$), suggesting that the long ventricular "pauses" associated with atrial fibrillation can be attributed to scaling of the maximal atrial cycle lengths. Abbreviations as in Figure 3.

discrete "levels" in the interval tachogram (a plot of RR interval against consecutive beat number) corresponding to a set of evenly spaced modes in the interval histogram. Soderstrom sought to explain the ordered structure of these modes as being determined by multiples of the refractory period of the AV node, which he was unable to measure. However, subsequent work (8,10,12) failed to discern any such structure in the patterns of the ventricular response. In particular, Bootsma (12) and Meijler (24) and their coworkers, using RR interval analysis alone, concluded that the AV node, other than scaling the atrial impulses, did not play a primary role in determining the irregularity of the ventricular response to atrial fibrillation.

Present study. We found several descriptors of frequency distribution histograms, including unimodality and the shape of the right-hand tail, to be comparable for both atrial and ventricular interval distributions in a group of 12 patients with atrial fibrillation. The predictability of both the ventricular variability and the maximal RR interval as scaled functions of the corresponding atrial measures adds to previous data derived from RR interval analysis alone, suggesting that the nature of the distribution of the RR intervals is primarily a consequence of the nature of the distribution of the AA intervals (10,12,23,24). This predictability would support the suggestion that the role of the AV node during atrial fibrillation is predominantly confined to that of scaling the atrial activity. These relations were also observed in six patients with atrial fibrillation and conduction over an accessory AV pathway, providing additional evidence that the irregularity and maximal intervals of the ventricular response to atrial fibrillation can be accounted for by the irregularity and maximal intervals of the atrial activity without assuming any special properties (other than scaling) of the AV conduction pathway.

How the AV node scales the rapid atrial activity to a slower ventricular response is unclear. Several interesting explanations have been proposed (4,14,33), but conclusive evidence in support of one specific mechanism has yet to be presented (34). Our study was confined to quantifying the relation between distributions of large numbers of atrial and ventricular events, and the exact mechanism of AV pathway function was not investigated.

Similarities between the AV node and accessory AV pathways during atrial fibrillation. The phenomenon of concealed conduction, long established as a mechanism of AV node behavior during atrial fibrillation (3–5,7), also has been demonstrated to affect the behavior of the accessory AV pathway (19). In addition, rate-dependent or "decremental" conduction over accessory pathways has been described (35,36). Recent work by Milstein et al. (20) showed that, when matched for anterograde effective refractory periods of their respective AV pathways, quantitative measures of the ventricular response to atrial fibrillation in patients with Wolff-Parkinson-White syndrome and those with enhanced AV node conduction did not significantly differ. The studies of Bauernfeind et al. (37) and Jackman et al. (38) indicate that enhanced AV node conduction represents one end of a continuous spectrum of normal AV node behavior. Therefore, it is not surprising that, when faced with rapid multiple inputs during atrial fibrillation, the AV node and the accessory AV pathway have qualitatively similar effects on the ventricular response.

Effect of drugs. Our data do not directly address the effects of negative dromotropic medications on either atrial or ventricular rate and variability. Administering a variety of dromotropic agents directly to the AV node of the dog, Billette et al. (17) observed that the variability of the RR interval distribution increased with the mean RR interval. Escudero et al. (13) found no significant differences in the degree of ventricular irregularity (measured as the coefficient of variation) among patients with a wide range of ventricular rates regardless of digitalis treatment. It appears that dromotropic medications do not change the manner in which the AV conduction pathway consistently scales the atrial variability to the ventricular variability. These results suggest that the effect of dromotropic interventions is limited to one or both of two possibilities: 1) a change in the rate or variability, or both, of the atrial activity itself, and 2) a change in the scaling ability of the AV conduction pathway (39).

Role of the AV node in "exclusive" conduction over an accessory AV pathway. A potential limitation of this study was the question of interaction between the AV node and the accessory pathway in patients with Wolff-Parkinson-White syndrome. The possibility exists that some AV node conduction did occur in these patients. However, the analysis of

the ventricular response to atrial fibrillation with conduction over an accessory AV pathway was limited to those patients whose QRS complexes appeared consistently fully pre-excited, reflecting an absence of fusion, and hence the absence of any evident contribution to the ventricular response by the AV node. Under these conditions, repetitive retrograde activation of the His bundle by way of the accessory pathway might preclude the AV node from participating in any AV conduction (15,40,41).

Determining the input/output of the AV conduction pathway. The R wave of the surface ECG lead identified the output of the AV conduction pathway, but determining the actual input to the pathway was more difficult. In addition to the probable existence of multiple inputs to both the AV node (3,42,43) and the accessory AV pathway (44), atrial activity recorded in the high right atrium is not necessarily similar to activity simultaneously recorded at other atrial sites (25). The effect of multiple inputs impinging on the AV conduction pathway is unclear; if the inputs respond independently with no consistent phase relation, then the apparent input rate to the conduction pathway would be much more rapid than measured elsewhere in the atrium (3). If, however, the separate inputs are not electrically independent, the effect of multiple inputs is less clear. The effects of multiple inputs to the AV node or accessory pathway would contribute to the behavior of the conducting pathway during atrial fibrillation and would be reflected in the statistical behavior of the output (i.e., the RR intervals). Thus, the complication introduced by the multiple inputs was inseparable from other factors determining the behavior of the AV conduction pathway and could not be examined mechanistically in this study.

Activity recorded in the high right atrium during atrial fibrillation is not an instantaneous measure of the input to the AV conduction pathway. However, there is no reason to believe that, over long periods of time, the statistical nature of atrial activation intervals would differ between the high right atrium and sites closer to the atrial insertion of the AV conduction pathway because of the random nature of atrial fibrillation (3,10,14,45). Therefore, for the purposes of this study, the assumption was made that over a period of minutes the statistical behavior of activation intervals measured in the high right atrium would provide an unbiased estimate of the statistical behavior of activation intervals closer to the AV conduction pathway.

Conclusions. This study adds direct observation of atrial events to a body of data that suggests that the irregularity of the ventricular response to atrial fibrillation is primarily a consequence of the irregularity inherent in the atrial activity. Ventricular variability and maximal RR intervals were related to atrial variability and maximal AA intervals by the scaling factor, and both the atrial and ventricular interval histograms had shapes similar to the Poisson distribution. This relation was observed in patients with and without accessory AV pathways, further supporting the conclusion that no special properties of the AV node need to be considered to account for the nature of the RR interval distribution.

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